

REPRODUCTIVE SYSTEM

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- D3.1.13 Control of the developmental changes of puberty by gonadotropin-releasing hormone and steroid sex hormones
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PUBERTY

Puberty describes the sequence of developmental changes that occurs within organisms to achieve sexual maturity. The changes will differ within males and females but both sexes follow a set sequence of events.

- **Gonadotropin releasing hormone** (GnRH) is released from the hypothalamus and acts on the pituitary gland to trigger the secretion of both follicle stimulating hormone (FSH) and luteinising hormone (LH)
- **FSH** and **LH** are transported via the blood to act on the gonads (testes in males and ovaries in females)

In males, FSH acts on the testes to trigger the activation of spermatogenesis, while LH is responsible for the production of **testosterone** – which promotes the development of secondary sex characteristics in males.

In females, FSH acts on the ovaries to trigger follicular development and the production of **oestrogen**, while LH is responsible for ovulation and involved in **progesterone** release. Oestrogen and progesterone are both involved in the monthly menstrual cycle and the development of secondary sex characteristics in females.

GAMETOGENESIS

Gametogenesis is the process by which germline cells undergo division and differentiation to form haploid sex cells. Females undergo **oogenesis** and males undergo **spermatogenesis**, but the process is similar in both and involves the following steps:

- Multiple mitotic divisions and cell growth
- Two meiotic divisions (i.e. meiosis I and II)
- Differentiation to form functioning gametes

Key differences exist between the two processes, specifically in the number of gametes produced, the size of the gamete (i.e. amount of cytoplasm) and the timing of the onset and duration of the process (i.e. it is either continuous or staggered).

Spermatogenesis	Oogenesis
Produces sperm in the testes	Produces ova (eggs) in the ovaries
Four gametes are produced by meiosis	One ovum is produced (plus polar bodies)
All gametes have equal amounts of cytoplasm	Unequal cytokinesis results in large ovum
Sperm is produced from puberty and continues until death (lifelong) – levels reduced with age	Egg production begins prenatally, continues via the menstrual cycle and ends with menopause

FERTILISATION

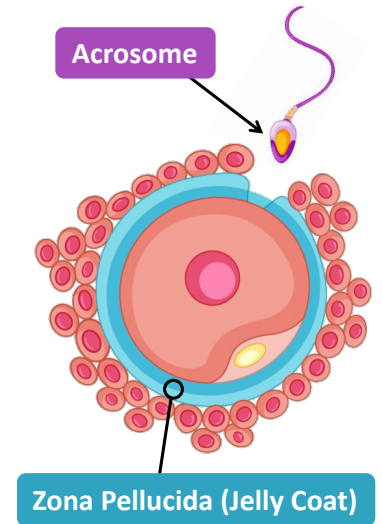
Fertilisation is the process whereby egg and sperm fuse together to form a diploid **zygote**. Successful fertilisation must involve two key processes:

Acrosome reaction:

The head of a sperm contains an **acrosome cap** with digestive enzymes. The cap fuses with the jelly coat of the egg and the enzymes soften the glycoprotein matrix, allowing a sperm to penetrate the egg membrane.

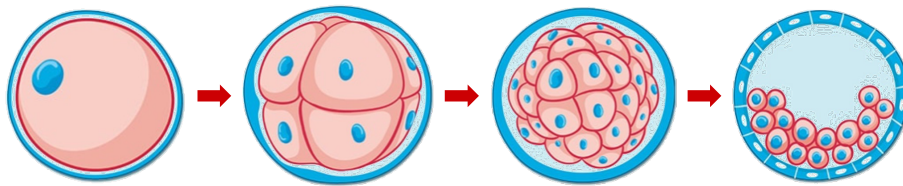
Cortical reaction:

Eggs contains **cortical granules** that release enzymes following sperm penetration. The enzymes destroy sperm binding sites and thicken the jelly coat – this prevents other sperm from penetrating (**polyspermy**).



IMPLANTATION

Following fertilisation, a diploid zygote undergoes several mitotic divisions to form a solid ball of cells called a **morula**. As the morula continues to divide, it undergoes differentiation to form a **blastocyst** composed of three distinct sections: an *inner cell mass* (forms the embryo), an outer layer of cells called the *trophoblast* (this develops into the placenta) and a fluid filled cavity called the *blastocoele*. The blastocyst travels via the oviduct to the uterus and implants in the endometrium, where it gains nutrition as it forms into an **embryo**.

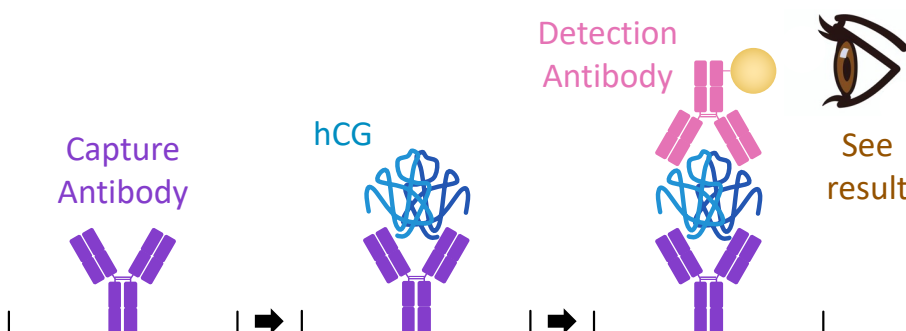


The dividing cells in a morula do not undergo G₁ and G₂ so the cells get smaller in size

PREGNANCY

When a blastocyst implants in the endometrial lining it will begin to secrete **human chorionic gonadotropin** (hCG). This hormone maintains the corpus luteum in the ovary and prevents its degradation. Consequently, the corpus luteum can continue to produce both oestrogen and progesterone, preserving the endometrium in which the blastocyst is implanted. The levels of hCG are maintained for 8–10 weeks while the placenta is being developed, then the placenta takes responsibility for hormone secretion and support of the embryo.

During early pregnancy, the presence of hCG in urine can be detected using **monoclonal antibodies**. These antibodies are made by fusing a plasma cell to a tumour cell to create an immortal line of hybridoma cells that mass produce antibodies specific to a target antigen (such as hCG). A standard pregnancy test contains two anti-hCG antibodies – one immobilised and one fixed to a colour-changing enzyme. When a pregnant woman urinates on the pregnancy test, both antibodies will form a complex with hCG and produce a band.

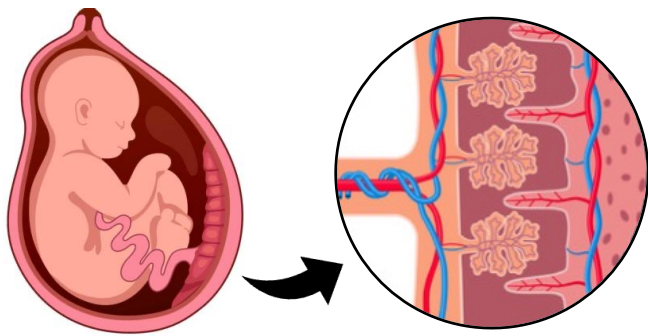


Pregnancy Testing:

Pregnancy can be tested using an **enzyme-linked immunosorbent assay** (ELISA) to detect the presence of hCG in urine

PLACENTA

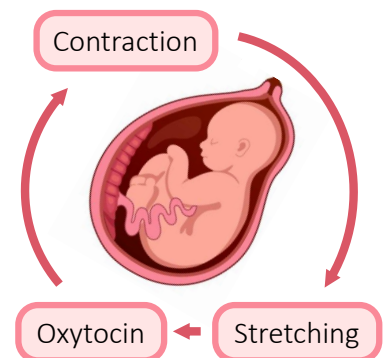
The placenta develops during pregnancy and functions as the life support system for the foetus. It secretes hormones to maintain pregnancy and facilitates the exchange of materials between the mother and foetus. Maternal blood pools from open-ended arterioles into internal cavities within the placenta. **Chorionic villi** from the foetus extend into these pools of blood and mediate the exchange of materials. Substances are transported via an **umbilical cord** connecting the placenta to the foetus. The foetus will absorb substances such as oxygen, nutrients, vitamins, antibodies and water from the maternal blood. Foetal products – such as carbon dioxide, urea and certain hormones – are transferred from a foetus and into the maternal blood. The placenta also secretes **oestrogen** and **progesterone**. Oestrogen functions to prepare for the process of childbirth by stimulating the growth of the uterine muscles and developing the mammary glands, while the progesterone prevents premature labour by maintaining the endometrium and inhibiting oxytocin release.



The increased surface area of the chorionic villi within the placenta functions to optimise the exchange of materials between the placenta and the foetus, which allows the foetus to be retained in the uterus to a much later stage of development than mammals lacking a placenta

CHILDBIRTH

Towards the end of a pregnancy, the progesterone levels start to drop. When the foetus has grown to a certain size in the womb, it will cause stretching of the uterine walls. This will trigger the release of **oxytocin** from the posterior lobe of the pituitary gland. Oxytocin will stimulate the uterine muscles to contract, resulting in even more stretching and further oxytocin release (**positive feedback**). The uterine contractions will lead to childbirth, which will finally remove the stretching stimuli.



HORMONE REPLACEMENT THERAPY

A woman remains potentially fertile until the onset of **menopause** (the permanent cessation of menstrual periods). Menopause results in the decreased production of the female sex hormones and causes various symptoms, including hot flashes and vaginal dryness. Menopausal women can be prescribed treatments of the sex hormones as a way of mitigating these symptoms. Early epidemiological studies suggested that the hormone replacement therapies reduced the risk of coronary heart disease (oestrogen can lower the blood cholesterol levels and reduce inflammation). However recent studies suggest these therapies may increase the risk of heart disease, breast cancer and stroke. There are several explanations for the different findings:

- The earlier studies relied on observational data, while later studies used randomised controlled trials
- The observational studies only focused on women in their early menopausal years (restricted sample)

Moreover, the correlation between hormone replacement therapies and a decreased incidence of coronary heart disease is not actually a cause-and-effect relationship. Hormone replacement therapy patients tend to have a higher socioeconomic status (they are wealthier), which affords them better access to vital health care. It is the socioeconomic status that has a causal relationship with lower risks of coronary heart disease.

Remember: Correlation does not equal causation (correlations only suggest associations, not causalities).